Chapter 5

FETAL PALEOPATHOLOGY

AN IMPOSSIBLE DISCIPLINE?

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This chapter introduces the concept of fetal paleopathology in archaeological material, highlighting the limitations and potential of such research to inform us about the lives of mothers and their babies in the past. Problems with preservation and recognizing lesions in such tiny skeletal remains are discussed, before potential new sources of research are highlighted.

Today, an estimated four million babies die each year worldwide within the first month of life (neonatal period), with the first day being the most critical period, and two-thirds of these babies dying within a week. The most common causes of death are prematurity, sepsis and pneumonia, asphyxia, tetanus, and diarrhea. None of these conditions are traditionally recognized in skeletal remains, and of the 14 percent who died of "other" causes, only 7 percent were from congenital conditions. These figures relate to death rates in industrialized countries that have clear social and economic divides, with children from poor backgrounds most at risk (Lawn et al. 2005). Other factors influencing neonatal mortality include low birth weight, complications during delivery, limited access to specialist care, maternal anemia, syphilis, and fever (Black et al. 2010).

But we should be cautious about directly transferring these data onto societies of different cultures, levels of development, and economies, especially in the past. Clinical studies have consistently identified a higher neonatal mortality rate for males, suggesting that females are more buffered against environmental insults in the womb and cope better with the stress of childbirth (Bekedam et al. 2002). While the precise mechanisms behind this phenomenon are still unknown, differential treatment where boys are favored can reverse this survivability pattern (McMillen 1979; Lawn et al. 2005). Application of neonatal and postneonatal death ratios may be used to explore whether children were dying from endogenous or exogenous causes (Lewis and Gowland 2007), but our imprecise age divisions make it a crude way of assessing perinatal causes of death.

Training in non-adult osteology has improved over the past decade, advancing an area of research that was previously underdeveloped. However, the identification of fetal remains and familiarity with their normal anatomy is still limited and may result in many subtle pathological lesions going unnoticed. Given all of these issues, it is understandable that paleopathologists rarely choose to study perinates, and a review of their potential to provide information on disease and trauma is long overdue.

Prenatal Pathology

The fetus is most vulnerable to environmental disruption during periods of rapid cell differentiation, particularly during the first two weeks of development when disruptive agents (teratogens) can result in spontaneous abortion of the embryo (Moore 1988). Skeletal development begins between eight to twelve weeks with the formation of the skeletal matrix, followed by intramembranous (in membrane) ossification of the clavicle, mandible, and bones of the skull vault. Endochondral (in cartilage) mineralization forms the rest of the skeletal structure with the ossification of the appendicular skeleton, ilium, and scapula by sixteen weeks, metacarpals and tarsals by sixteen to twenty gestational weeks (Krakow et al. 2009). Successful ossification relies on a good maternal oxygen supply through the bloodstream during fetal development (Waldron 2009). The homeobox (Hox) genes regulate the differentiation process of the skeleton into limb or spinal elements, and mutations in these genes are responsible for malformations during embryonic development (Scheuer and Black 2000: 172).

Infections can spread to the fetus via the placenta. The placenta is highly vascular, and any breach of its integrity may lead to large amounts of a pathogen reaching the fetus. Pathogens may also spread through the umbilical vein, through ingestion of infected amniotic fluid, or during birth with exposure to an infected birth canal or maternal fluids (Zeichner and Plotkin 1988). The fetus may suffer damage or disruption to their developing cells, or may mount an autoimmune response, but because the fetus has a reduced inflammatory reaction to infection (Holt and Jones 2000), skeletal signs of disease are limited before birth. Rubella, smallpox, tuberculosis, syphilis, leprosy, chickenpox, mumps, measles, and scarlet fever can all be transmitted transplancentally (Lorin et al. 1983; Naeye and Blanc 1965; Al-Qattan and Thomson 1995) and may be evident on the perinatal skeleton, either through signature (pathognomonic) signs, or generalized new bone formation. While exposure to influenza is responsible for many congenital defects, leprosy and syphilis may instead cause early spontaneous abortion or, if transmitted toward the end of the third trimester, remain latent until later infancy and childhood (Dorfman and Glaser 1990, Melsom et al. 1982). While congenital syphilis may result in characteristic notches on the unerupted deciduous upper incisors on radiograph (Hutchinson 1857), cranial deformities caused by malaria or maternal smoking (Lampl 2003) are all but impossible to identify on thin and unfused perinatal cranial bones.

Enigmatic Skeletal Lesions

New Bone Formation

Traditionally, paleopathologists interpret gray bone deposits (fiber bone) on the outer bone surface as a sign of trauma or infection. For the perinatal paleopathologist, things are less straightforward. P. de Silva and colleagues (2003) warned clinicians against misdiagnosing what they termed "physiological" periostitis as abuse in infants. Periostitis is a term used to denote inflammation (or inflammitis) of the fibrous sheath (periosteum) surrounding the bones in life. They noted symmetrical new bone formation on the long bones, especially the femora, humeri, and tibiae in babies aged one to six months old. While the new bone was usually concentric, the tibia was most commonly affected on the medial aspect, and in most cases, bone formation was confined to the long bone shaft (or diaphysis).

Charles Shopfner (1966) examined the radiographic appearance of the long bones of 335 healthy premature and full-term infants, and he noted periosteal new bone in 35 percent of cases. The bone deposits were thick, but not multilayered, and appeared on radiograph as double contours before they became incorporated into the underlying bone surface. K. Gleser (1949) warned that increased formation and mineralization of the long bones during the normal growth process might mimic pathological features in a two- to five-month-old month infant, when signs of congenital syphilis, scurvy, and rickets may be suspected. For the paleopathologist examining dry bone, perinates with widespread new bone formation on the cranium, long bones, and ilia are common findings, but we are ill equipped to determine if this indicates one of the many infections that may be responsible for neonatal death or if this signals the child was experiencing a growth "spurt" when they died. Common sense may dictate that a child on the brink of death is unlikely to be undergoing rapid growth, but this does not account for accidental deaths or deaths that occur shortly after a growth spurt but before the bone can remodel.

The bone turnover rate in neonates has been estimated to be high before birth and in the first forty-eight hours of life, and greater in the preterm neonate than full-term babies, with bone turnover rates of infants being several times higher than in adults (Mora et al. 1997). While the extent of bone turnover in perinates may be difficult to quantify, remodeling of the femur, for example, significantly changes bone density within the first six months. Hence, any new bone formation in perinates will be rapid, with several days needed for the newly deposited organic matrix to be mineralized (Rauch and Schoenau 2002). It would also be reasonable to expect any trauma or inflammation experienced during the birth process to be remodeled within six months of the child's life, making identification of lesions in the perinates all the more crucial.

In some cases, localized or profuse new bone deposits that are gray may point to specific conditions. Infantile cortical hyperostosis (or Caffey disease) is an inflammatory disorder of unknown etiology causing profuse new bone formation on the long bones and mandible that heal spontaneously (Caffey and Silverman 1945). A. Alduc-Le Bagousse and J. Blondiaux (2001) suggested a possible fetal case, found within the abdomen of a female in Lisieux, France. All the surviving long bones have profuse new bone formation, and the tibiae and cranium are most severely affected. Layers of new bone are often identified on the internal cranial surfaces of perinatal material, confined to the occipital. As this part of the cranium is undergoing rapid growth after birth, many of these lesions are likely part of the normal bone remodeling process (Lewis 2004), but they may also be signs of inflammation or calcified blood pools (hematomas).

J.P. Crozer Griffith (1919) states that perinatal intracranial hemorrhage occurred in 15 percent of premature births and may result from cerebral palsy. Birth trauma may cause localized bleeding and, if a child were laid on their back, then blood or pus would pool to the occipital area (Mitchell 2006). A. González Martin and colleagues (1997) raised the possibility that the irregular and porotic appearance of the pars basilaris (the bone that forms part of the base of the skull) seen in children from birth to six years was potentially pathological, calling for more research into this area. Without more detailed information on the pattern and timing of perinatal growth in individual skeletal elements, we may never be able to untangle these issues.

Lytic Cranial Lesions

Tania Kausmally and Rachel Ives (2007) highlight problems with the interpretation of destructive (or lytic) lesions in 7.4 percent (seven out of ninety-four) of perinates from postmedieval London. The lesions caused holes in the cranium but only occurred on a few of the perinates from the same site and context, suggesting postmortem damage was not to blame. Kausmally and Ives identified several possible causes including cancer—for example, infantile chordoma, infantile myofibromatosis, Langerhans cell histiocytosis (LCH), and tuberculosis (TB). As the lesions were too common to support very rare cancerous conditions, they favor TB or LCH as a cause.

John Caffey (1978: 32) had previously noted these lesions, calling them "lacunar skull" and describing them as most marked on the parietal and frontal bones. He linked them to spina bifida, hydrocephalus (cranial enlargement due to "water on the brain"), meningocele, and meningioencephocele (protrusion of the brain lining through the cranial vault), but he also saw them on the radiographs of apparently health newborns. As the lytic lesions tended to heal spontaneously, Caffey considered them the result of delayed development of the membrane that eventually forms the cranial vault. Sheila Mendonça de Souza and colleagues (2008) noted destructive lesions on the skull of a six-month-old Peruvian mummy that were accompanied by new bone formation on the internal surface of the cranium, active bone formation on the frontal and parietal bones, and a flattened occipital. They suggest cranial modification through head binding, followed by bone death and secondary infection as the cause.

Potential Perinatal Paleopathology: Infections

Rubella

Congenital rubella results from transmission of the rubella virus during the first trimester and would have had serious consequences for the survival of a newborn in the past. Pregnancies ending in a spontaneous abortion or stillbirth would result in perinates entering the skeletal record without pathological changes. Those born alive suffer congestive heart failure, low birth weight, and difficulty in feeding, along with deafness and cerebral palsy in older child (Cooper et al. 1969). Arnold Rudolf and colleagues report that 45.3 percent of perinates with exposure to maternal rubella display bone lesions between the ages of one to eight weeks and that 76 percent of the thirty-four perinates are male. Osseous changes include wide radiolucent bands and "beaklike projections" at the ends of the bone shafts (metaphyses) during healing, coupled with enlarged anterior fontanelles at the areas where the cranial sutures meet (Rudolf et al. 1965: 430).

The rubella virus's ability to inhibit cell multiplication of bone and fiber forming cells (i.e., osteoblasts and fibroblasts) and other tissues in the body is well known (Naeye and Blanc 1965; Reed 1969). These poorly defined zones of calcification are similar to what paleopathologists might see in children with rickets or congenital syphilis. However, once virus excretion has ceased, bone lesions can disappear in several months (Sekeles and Ornoy 1975). While slightly older than perinates, three infants aged between three and six months were identified with unusually large fontanelles in South Africa dating to the twentieth century, leading M. Steyn and colleagues (2002) to suggest rubella as a possible differential diagnosis. Although rubella lesions are rare and transient in the perinatal period, it should be considered a differential diagnosis in early rickets cases.

Neonatal Osteomyelitis

Neonatal osteomyelitis, an abscess-forming infection affecting multiple bones, was a common cause of death in the past (Trueta 1959) but has yet to be identified in perinates from archaeological contexts. Clinically, multiple bone and joint involvement occurs in 41 percent and 70 percent of cases, respectively (Weissberg et al. 1974), assisted by the presence of open transphyseal vessels that allow the spread of infection across the growth plate (Trueta 1959). John Ogden (1979) describes abscess and new sheath (sequestrum) formation in the first few days after birth due to a blood-born spread of infection and from a localized infection such as a burn. The humerus in the upper arm and the knee are recognized as common sites of infection, and radiographs of these areas may reveal abscesses inside the bone, while enlarged nutrient foramina may suggest involvement of transphyseal vessels.

Sheaths of new bone should be readily identified in the perinatal skeleton, but long bone epiphyses (the growing end plates) are not ossified at birth, and smooth-based localized lesions on the metaphyseal surface caused by abscess may be more difficult to distinguish from postmortem damage or a normal undulating surface. Caution is needed when using clinical cases to reference diseases from the pre-antibiotic era. Ogden's (1979) neonates had chemotherapy that may have allowed development of chronic lesions where more rapid death would normally occur. How common neonatal osteomyelitis was in the past is difficult to judge as the emergence of penicillin-resistant *Staphylococcus* meant a resurgence of osteomyelitis in a more virulent form and a higher number of neonatal cases from the 1950s to 1970s (Gilmour 1962).

Early Onset Congenital Syphilis

Congenital syphilis develops in the fetus secondary to venereal syphilis in the mother. The causative organism, *Treponema pallidum*, can be transmitted as early as the ninth week of gestation. The pathogen enters the fetal bloodstream and spreads to almost every bone in the body. Toxins released from dead microorganisms may invoke an allergic response and uterine contractions in the mother, resulting in fetal death and spontaneous abortion (17 percent) in the first half of the pregnancy (Genç and Ledger 2000). These tiny skeletal remains, if they survive into the archaeological record, will show no signs of disease. At term, a child may be stillborn (23 percent), premature, weak, and sickly, or between 39 and 66 percent may appear perfectly healthy (Harman 1917; Hollier and Cox 1998). About 21 percent of the latter will go on to display signs of infection around two years of age ("late congenital syphilis") (Harman 1917).

A trio of skeletal lesions occurring together is indicative of congenital syphilis in the perinate: joint disruption (osteochondritis), osteomyelitis, and profuse new bone formation (Caffey 1939). György Pálfi and colleagues (1992) describe a seven-month-old fetus recovered from the abdomen of a woman from Costebelle, France. The fetus displayed profuse new bone on the long bones, maxilla, ribs, and cranial vault; possible destructive lesions on the parietal bones; and a characteristic Wimberger's sign (thick dark band) on a radiograph. Convinced they had a case of congenital syphilis, the authors argued that the mother was in the early stages of syphilis, where clinical evidence suggests nearly all pregnancies will involve the spread of infection to the developing child. More recently, aDNA analysis (Montiel et al. 2012) confirmed syphilis in two perinates from postmedieval Huelva, Spain (Malgosa et al. 1996). This was the first time DNA successfully identified a subspecies of the treponeme and may have been because of the abundance of spirochetes known to invade the bone cells of neonates (Montiel et al. 2012).

Potential Perinatal Paleopathology: Congenital Defects and Skeletal Dysplasia

The prenatal ossification process begins around eight to twelve weeks in utero and from this point, osteologists have the potential to recognize congenital defects in the skeleton. In particular, irregularities in the formation of bones and replacement of the fetal spinal cord, or notochord, mean we have the opportunity to identify lesions that may signal more serious soft tissue defects resulting in perinatal death (fig. 5.1). Bruce Anderson (1989) describes axial

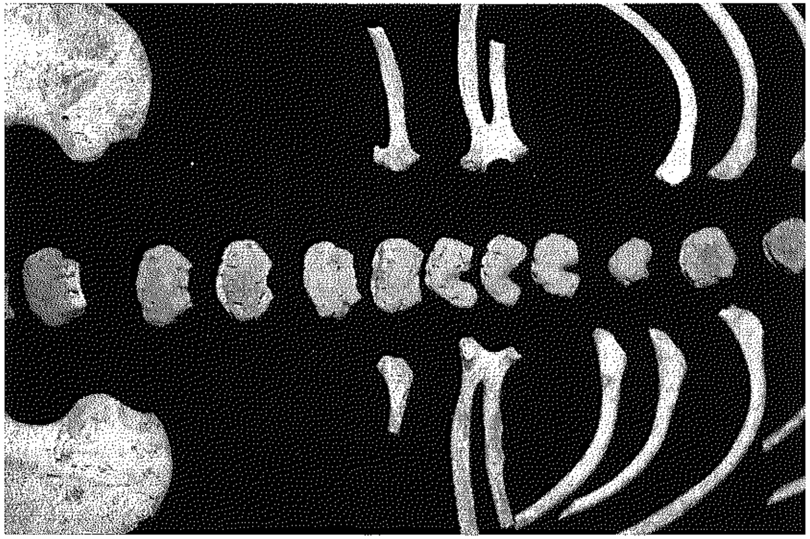

FIGURE 5.1. Congenital defects in the thorax of a thirty-nine-week-old perinate from St. Oswald's Priory in Gloucester, England. The poorly mineralized centra and fused ribs may signal a more serious soft tissue condition that led to the child's death. (Photo courtesy of author.)

congenital anomalies and a possible familial relationship in three neonates from Homol'ovi III, Arizona. The first child presented fused thoracic (chest) vertebrae and asymmetry of the maxilla and mandible with dental overcrowding, suggesting marked facial asymmetry. The second was a perinate with fused second and third cervical (neck) vertebrae and two mid-thoracic vertebrae, possibly indicating type 2 Klippel-Feil syndrome. The child also had a malformed rib. Finally, a younger perinate also demonstrated fusion (or non-separation) of the spinous processes of the second and third cervical vertebrae and the fourth and fifth thoracic vertebrae. M. Hinkes (1983) noted flared sternal ends at the front of ribs 1–8 in a neonate from the Grasshopper Pueblo, and Don Brothwell and R. Powers (2000) recorded merged ribs in a neonate from early medieval Lechlade, England. A tiny example of a fused radius and ulna in the arm (radio-ulnar synostosis) has been reported in a perinate from a double burial at El Molon, Spain (Lorrio et al. 2010).

Cleft, butterfly, and block vertebra, as well as anterior and posterior spina bifida, all have the potential to be identified in the perinate (Müller et al. 1986), and the presence of extra cervical ribs may reveal information about the potential cause of death. In a study of 318 perinates born in Utah between 2006 and 2009, Larissa Furtado and colleagues (2011) reported a significantly higher prevalence of cervical ribs in stillborns compared to live-born children who died within the first year (43 percent compared to 12 percent). They conclude that cervical ribs signal a disadvantageous fetal environment that leads to a greater likelihood of stillbirth with similar results presented elsewhere (Bots et al. 2011). This would require identification and careful examination of the neural arches for the seventh cervical vertebrae for facets. Although, Sue Black and Louise Scheuer (1997) note that cervical ribs themselves will not be found in children under the age of ten years, as they do not fully develop until fusion of the posterior arch to the body of the vertebrae.

More than 350 forms of skeletal dysplasia have the potential to be identified in perinatal remains. Differentiating between them is clinically problematic so is likely to be impossible in archaeological circumstances, although some features such as glabella bossing at the top of the nose, flattened nasal bridge, vertebral (centrum) morphology, and poor mineralization of the cranium and skeleton may signal dysplasia (Krakow et al. 2009). Osteofibrous dysplasia of the neonate, while infrequent, may present as an expansile lytic lesion at the midshaft of a single tibia causing bulging of the bone surface macroscopically and, potentially, a pathological fracture (Hindman et al. 1996). Mark Mooney and colleagues (1992) presented a series of fetal facial measurements that can be used to identify cleft lip and palate, defects that are hard to recognize in the tiny unfused perinatal maxillae. These include the premaxillary length and nasal opening length, which can theoretically be identified on dry bone, but techniques to test and apply this to archaeological material have yet to be developed. Although there is potential for more research to date, congenital skeletal defects are the most common form of pathology identified in archaeological perinates.

T. Sjøvold and colleagues (1974) identified a full-term fetus with multiple bony projections (osteochondromas) from St. Clement, Visby in Gotland, found within the abdominal area of a seventeen- to twenty-year-old female with the same condition. The deformities may have caused an obstruction leading to the death of the mother and child. Kenneth Bennett (1967) describes a case of multiple cranial suture fusion in a perinate from Utah that would have led to a cloverleaf deformity if the child had lived. Darcy Cope (2008) suggests the presence of a meningocele in the fragile cranial bones of a neonate from the Dakhleh Oasis, and a perinate with an even more severe cranial malformation (holoprosencephaly) (Tomasto-Cagigao 2011) buried in an urn in Palpa, Peru. The burial was normal except for the presence of another apparently normal neonate. Although E. Tomasto-Cagigao (2011) does not describe the rest of the skeleton, segmental errors in the spine are often associated with this cranial deformity (O'Rahilly et al. 1980, 1983).

The oldest archaeological case of a perinate with anencephaly (a fatal condition where the brain and the bones of the cranial vault fail to form) comes from an Egyptian catacomb in Hermopolis, built to house the mummies of sacred monkeys and ibises (Saint-Hillaire 1826). J. Christopher Dudar (2010) discusses a possible case of anencephaly in a child from Elmbank Cemetery in nineteenth-century Toronto with associated fused ribs. Detailed studies of the perinates from the Dakhleh Oasis, Egypt, have revealed a variety of remarkable cranial malformations (encephalocele, iniencephaly, and anencephly) (Cope 2008). Stevie Mathews (2008) provides a useful description of known anencephaly in perinates housed at the Smithsonian Institution in Washington, DC. Deformities included malformed sphenoid lesser and greater wings, lack of frontal and parietal bones with isolated orbital rims, deformed squama of the temporal bones, and early fusion of the elements with an elliptical rather than round tympanic ring (a small ring of bone that forms at the external opening of the ear).

A.L. East and J. Buikstra (2001) discuss an achondroplastic female from Elizabeth Mounds, Tennessee, with an in utero fetus they also suspected had achondroplasia based on metaphyseal flaring, disproportionately short limbs, and cranial and long bone measurements that were outside the normal range of the forty-six perinates they compared it to. This case has not been fully published, and the authors caution other dysplasias have yet to be ruled out. Arthur Keith (1913) provides a very useful comparison of the individual bones of an achondroplastic child against an unaffected child of the same age. The affected child displayed a reduced foramen magnum, absent suture mendosa on the developing occipital bone, premature fusion of the basioccipital suture, and short and broad wing of the pars lateralis. In the full-term child, the basiocciput and presphenoid fused limiting further expansion of the brain. Frontal bossing was evident because of the brain seeking compensatory space.

Down Syndrome

Trisomy 21 is the most common chromosomal abnormality among live-born infants and is related to increasing maternal age (e.g., thirty-five years). Today, Down syndrome is estimated to occur in one in seven hundred to one in a thousand births in the United States (Benacerraf 1996). The vast array of skeletal and dental features associated with this syndrome has always attracted the attention of paleopathologists, and while five suspected cases of Down have been identified, none of these are perinates. This may be because children with the syndrome tend to survive into older childhood, but given mothers of increasing age are more likely to suffer from complications at birth, we might expect some cases to enter our perinatal sample. Ernest Hook (1979) estimated 21 percent of trisomy 21 children died between midgestation and full-term. Sonographs of second-trimester fetuses reveal some skeletal features used to predict the presence of trisomy 21 at birth. These include a shortened femur and humerus when measured against the maximum width of the skull and in comparison to the population norm, a shortened iliac length, reduced limb bone length in comparison to the axial skeleton, and shortening of the middle phalanx of the fifth digit of the hand (Keeling et al. 1997; Stempfle et al. 1999). Unfortunately, these features require either an intact cadaver or a large enough series of well-preserved perinates to gauge the normal dimensions. Although hand phalanges ossify around twenty-four gestational weeks, they are so tiny they are rarely excavated, making measurement or an assessment of agenesis impossible. In addition, the short and stumpy fifth-finger phalanx, if recovered, may be misidentified as belonging to the foot. Clinically, the accuracy of these measurements in identifying Down syndrome is between 40 and 50 percent, only rising to 74 percent when the soft tissue nuchal fold of the neck is included (Benacerraf 1996).

Nevertheless, some features hold promise. Absence of the nasal bone is commonly reported in Down syndrome neonates (Dedick and Caffey 1953; Keeling et al. 1997; Otaño et al. 2002; Stempfle et al. 1999) and should ossify between fifteen and forty gestational weeks. L. Otaño and colleagues (2002) found absent nasal bones in three of five (60 percent) of Down syndrome fetuses compared to only 0.6 percent of non–Down syndrome cases. A related condition, trisomy 13, can result in malformations in the lumbosacral and thoracic spine, where small and irregular bones have been noted (Kjær et al. 1997). A fetus with triplody, a rare and lethal condition where there are three sets of chromosomes, can present with cranial base malformations including extra ossification centers, and fusion of two or more vertebral bodies, or a disproportion in the size of the cervical bodies. Another chromosomal disorder (aneuploidy) is a possible explanation for the cranial deformations identified in a thirty-eight- to forty-week perinate from Andover Road in Winchester, United Kingdom. The wing of the pars lateralis is bipartite, and the posterior condyle canal is in two halves (fig. 5.2). The thickened and regular edges suggest this was a congenital anomaly rather than a basilar linear fracture of the occipital (Falys 2010).

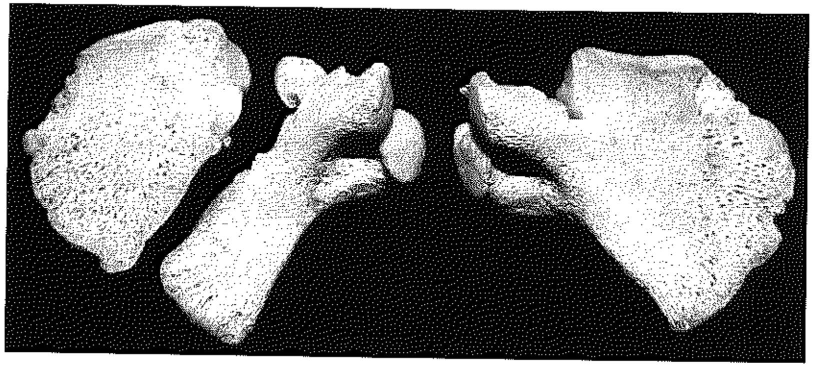

FIGURE 5.2. Possible aneuploidy in a thirty-eight- to forty-week-old perinate from Andover Road in Winchester, United Kingdom (reproduced with kind permission from Ceri Falys, TVAS).

Osteogenesis Imperfecta

Osteogenesis imperfecta (or "brittle bone disease," a fatal congenital condition leading to multiple fractures) is a probable diagnosis for a thirty-eight-week-old perinate from Dakhleh Oasis, Egypt, with severe bowing of all the surviving long bones and pathological fractures of the left ulna, femur, and tibia (Cope and Dupras 2011). The baby was buried on its side in contrast to the normal supine extended burials of other children in the cemetery. Although the basilar fragments of the skull were preserved, they did not show the malformations (for example, a triangular pars basilaris) identified in a forensic case from Guatemala (Lewis 2007: 107).

Potential Perinatal Paleopathology: Trauma

Perimortem cut marks on neonatal remains have been interpreted as indicating surgical removal (or an embryotomy). Only three cases have so far been identified; two are Romano-British examples from Poundbury Camp in Dorset and Yewden villa, Buckinghamshire. The Poundbury perinate was decapitated and has extensive cut marks throughout the long bones (Mays et al. 2012; Molleson and Cox 1988). A possible nineteenth-century embryotomy was identified in L'Aquila, Italy (Capasso et al. 2016), based on severe jumbling of the bones in a wrapped and mummified twenty-nine-week-old fetus.

Birth Injuries

Perinates who die shortly after birth are too young for us to assess any paralysis that may occur as the result of trauma during childbirth, but some fractures may be evident. In 1950s New York, 6 percent of all neonatal hospital admissions of newborns were for injuries sustained at birth (Montagu 1950). Andrew P. Dedick and John Caffey (1953) reported fractured clavicles in 1.2 percent of their 1,030 newborns; these were always unilateral and occurred more commonly on the left side. Caffey (1978) describes hematomas (cephalhematomas) on the ectocranial surface as the result of bruising during breech birth usually positioned away from midline sutures, a detail that may aid in their differentiation from meningocele.

Ossified hematomas may persist for months or years. Skull fractures as a result of birth injury present more of a challenge because of the fragile and fragmentary nature of the perinatal cranium. Linear and depressed fractures may not survive fragmentation in the ground, or if the child dies shortly after birth, perimortem fractures may be indistinguishable from postmortem breaks. Caffey (1978) also suggests that the maxilla is a frequent site for infection in the first few weeks of life because of birth trauma and may be visible as reactive new bone formation around the developing dental germs. Three published cases of possible birth injuries have been identified in paleopathology, involving a skull fracture in a thirty-eight-week perinate (Baxarias et al. 2010) and two cases of unilateral clavicular fractures (Brickley et al. 2006; Soren et al. 1995).

Conclusions

This chapter has reviewed the challenges and potential of examining perinatal remains from archaeological contexts in order to identify skeletal pathology. Although the majority of perinates likely died from infectious or innate conditions, identifying pathology based on new bone on subperiosteal and endocranial surfaces is problematic, as we have yet to develop criteria that allow us to distinguish pathological lesions from the normal growth process. Nevertheless, there has been increasing success in the identification of congenital malformations from archaeological contexts, with fifteen of the twenty-nine (52 percent) reported perinatal cases describing congenital disorders. While fetal remains recovered from the pelvic cavities of female graves hint at obstetric hazards, individual perinatal burials have the potential to tell us much about the health of the fertile maternal population, as well as the environmental factors that affect the survival of newborns. A review of the clinical literature has allowed for the identification of skeletal features that may help us recognize new conditions in the perinate, including rubella, Down syndrome, and other chromosomal disorders, and birth trauma and cleft palate, but further research is needed to understand the range of pathology that can be identified on these tiny remains. Appreciating how far we still have to go in our analyses will help us set the agenda for future studies.

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